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Chapter

Local Anesthetics in Odontology

Enrique Hernández-Cortez, Cecilia G. Sandoval Larios and Juan Carlos Flores-Carrillo

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Abstract

Pain has been a faithful companion of human beings and is a result of most of the dental procedures and illness; therefore a good control of dental pain is inevitable and feasible. The administration of local anesthetics has come to be the standard of care of dental profession. All local anesthetics are effective and high safety margin in all patients including childhood. The choice of using a local anesthetic depends on time of the surgical procedure, patient's medical history, and the interaction between local anesthetics and patient usual medications. The use of vasoconstrictors is priority in surgical/dental bleeding, but it must be used with caution in patients with cardiovascular disease to avoid a dental catastrophe. Dentists should be experts in dental-anesthetic techniques and in pharmacology of local anesthetics, since they are the most used medications in odontology. The accidental injection or a high absorption of local anesthetics in blood results in systemic toxicity. In such situation sedation, stunning, diplopia, sensory disturbances, disorientation, muscles spasm, respiratory depression, seizures, or cardiac arrest may be present; the dentist must immediately recognize this clinical complication to stablish an early treatment.

Keywords: local anesthetics, dental, local anesthesia, local analgesia, complications

1. Introduction

Local anesthetics (LAs) are the most used drugs by dentists. Actually, they are safer drugs. More than 40% or urgent dental procedures cause pain that needs and injection of LA. Therefore, dentists should be experts in dental-anesthetic techniques and in pharmacology of LA drugs. The safe administration of LA is the standard of care of dentists.

Pain has been a companion of human beings since their appearance on earth. Before local anesthetics, natural medicine was first used to relieve dental pain. Such medicine has evolved since ancient Egypt and Greek culture until the nineteenth century when LA developed. Actually, LAs are the most secure and effective drugs in medicine for pain prevention and treatment. There are no drugs more effective than LAs to avoid pain; no other drug prevents a nociceptive impulse from reaching the patient's brain, to finally be interpreted as pain. Whenever local anesthetics are administered near a sensitive nerve, it produces an adequate control pain for a limited time with the aids of been a reversible and temporary effect without harm of the anesthetized nervous structure.

The first registered dental anesthesia in history was in 1885 of the alveolar inferior nervous, applied by the medical surgeon William Stewart Halsted. The injected drug was a combination of cocaine and epinephrine [1]. In 1905, 2% procaine with epinephrine 1:50,000 was introduced, giving a quick access to dentists worldwide. Procaine, propoxycaine, and tetracaine were the most used LAs until the middle 1940s. Lidocaine was synthetized in Sweden in 1948 [2]. Articaine was synthetized in 1973 and introduced in dental clinic in 1976 and approved in Canada in 1984 and in the USA until 2000. Articaine has special characteristics of both amino amide and amino ester anesthetics. Its popularity has increased rapidly and is currently displacing the use of lidocaine in dental anesthesia [3].

Annually, a dentist in Canada applies 1800 LA cartridges, while in the USA, more than 300 million of cartridges are administered each year. Therefore, dentists should be experts in pharmacology, complications, and secondary effects of LA. In daily practice there are few complications related to LA, owing its security and the relative low doses that are applied. Nonetheless, it is necessary to consider possible complications, to detect it as soon as possible.

2. Pharmacology of LA

The synthetic LA has a common chemical structure, constituted by an aromatic ring, a hydrocarbon channel, and an amino group; the hydrocarbon channel and aromatic ring are joined by an ester or amide bond. Most of LAs are tertiary amines. The lipophilic portion is the biggest of the molecule. The aromatic portion proceeds from benzoic acid, aniline, or thiophene (articaine) and is the lipophilic portion. This portion is responsible for the affinity of nerve cells. The hydrophilic portion is an amino ethanol or acetic acid derivative and is responsible of water solubility and diffusion across tissues. LAs are amphipathic; that is, they have lipophilic and hydrophilic characteristics at their opposite ends of their molecules (**Figure 1**) [4]. Amides are the most common molecules; procaine is the prototype of this group and benzocaine for topic application. The minimal concentration of LA to block the conduction of a nociceptive impulse is named potency. The therapeutic value of the drug in terms of efficacy and tolerability is called toxicity. The ability of the drug to reach tissues far from the site of administration of LA is called diffusion. Time between the action of LA and the metabolism of its compounds is named time of action [5].

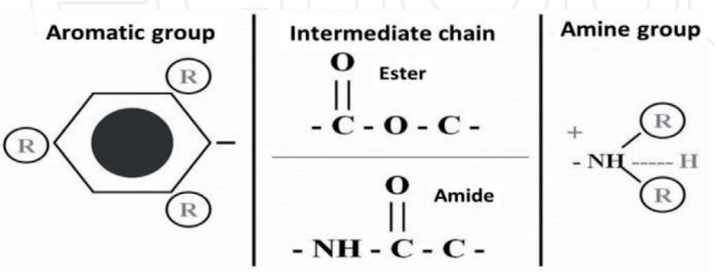

Figure 1.
Local anesthetic molecule.

3. Mechanism of action of LA

The type of nervous blockade produced by LA is named non-depolarizing nervous block. Local anesthetics block sodium channels permeability, they selectively

inhibit the maximum permeability to sodium, whose values usually are five to six times greater than the necessary minimum to conduct the impulse, when this value fail, and the nerve block occurs. In other words, local anesthesia is induced when the spread of action potential is inhibited, so a painful sensation cannot be transmitted from the site of origin to the brain. Or, LA alter the mechanism to sodium ions to gain access to the axoplasm nerve. The nerve membrane stays in a polarized state due to the impossibility of ionic movement responsible of action potential. LA blocks the entrance of sodium ions into their channels in the nerve cellular membrane. The permeability to sodium is necessary to generate a new action potential to transmit nerve impulses to the brain [6]. The sequence of action mechanism proposed for LA is described in **Table 1**.

LAs are classified into two large groups depending on its chemical bond: amides or ester; the latter are almost disappearing in dentistry. The ester LAs are easily hydrolyzed in aqueous solution, while amide bond LAs are relatively resistant to hydrolysis. The most important factors that affect onset and duration of action of LA are tissue pH, drug pKa, diffusion time from the tip of the needle to nerve, nerve morphology, drug concentration, and drug solubility in lipids [7]. The most important of the abovementioned factors are tissue pH and drug pKa. The pH may be reduced in infection sites, so the result would be a delay in the LA onset. In clinics, the amides pK is similar, with the exception of bupivacaine that has a slightly greater pK and an onset time more prolonged. The proximity of the anesthetic to the nerve is another factor that influences the action time of LA; a very clear example is the Gow-Gates blockade, very slow in its installation. The nerve morphology is also an important factor, which thin fibers are quickly anesthetized compared to thicker ones. The duration of action depends on the depth at which the drug may be blocking the sodium channels in the nerve membrane. If the LA causes vasodilatation, it allows a more or less rapid diffusion in the site of action and therefore a short duration of action, especially if the drug is administered alone. This diffusion may be reduced with the aid of vasoconstrictors like epinephrine, although bupivacaine is the only LA that has a prolonged action duration [8]. The two most important factors involved in the action of LA are the drug diffusion across the nerve sheath and the bind to the receptor in the ionic channel; a lipid-soluble free base and without electric charge is responsible of the diffusion across the nerve sheath.

The biotransformation of amides occurs mainly in the liver, although prilocaine is metabolized in plasma and eliminated in the kidney. One of its metabolites could be the origin of methemoglobinemia [9]. The ester LAs are biotransformed in plasma by enzymes called cholinesterase or pseudocholinesterase produced in the liver [10].

1. Displacement of sodium ions from the sodium channel receptor
2. Binding of the LA molecule to the receptor
3. Block of the sodium channel
4. Decreased sodium conduction
5. Depressed velocity of electric depolarization
6. Failed to teach the value of umbral potential
7. Absence of propagated action potentials
8. Conduction block

Table 1.
Sequence of mechanisms of LA to produce conduction block.

Specific action
Reversible action
Quickly onset action
Adequate duration of action
Active, injected, or topically administered
Non-irritating
Non-permanent harm
No systemic toxicity when properly used
High therapeutic ratio
Chemically stable
Long half-life
Ability to combine with other agents without losing their properties
Sterilizable without losing its properties
Slight allergenic

Table 2.
Important properties of LA.

Despite the great advances in the field or anesthetics in dentistry, the ideal agent do not exist; however, new ALs are increasingly safer. Some of its most important properties are listed in **Table 2**.

4. Most commonly used LA in dentistry

4.1 Lidocaine

This is the most common used LA in dentistry since its introduction in 1948 and now considered the gold standard for clinical use. It was the drug that displaced older anesthetics like procaine (Novocain). It has fast onset between 2 and 3 minutes with a longer and profound anesthesia. It is available in various concentrations; the most used are 1 and 2% cartridges with 1:100,000 and 1:50,000 epinephrine. Using lidocaine without a vasoconstrictor is rare in dentistry because of rapid vasodilation and high plasma concentrations as well as higher adverse reaction. For patients who are sensitive to epinephrine, they should limit the amount to a maximum of two cartridges per procedure. Thus, it is true for hypertensive patients, patients with coronary heart disease, and the elderly. Lidocaine combined with 1:50,000 epinephrine is commonly used in active dental bleeding because of diminishing up to 50%. It gives a pulpal anesthesia for up to 90 minutes and sometimes may be comparable to articaine (3–5 hours). Today, lidocaine is referred to local anesthetics toxicity. The maximum recommended dose in combination with epinephrine is 7 mg/kg for an adult or children over 15 kg, not exceeding 500 mg. Lidocaine alone is limited to 4.4 mg/kg, not exceeding 300 mg [11].

4.2 Mepivacaine

This is an amide type LA, available only in cartridge formed at concentration of 2 and 3%. Mepivacaine combined with a vasoconstrictor in 2% concentration. It is ideal for patients with an absolute contraindication to receive vasoconstrictor.

A maximum recommended dose in adult or children is 4.4 mg/kg without exceeding 300 mg a day. Simple mepivacaine at 3% gives anesthesia for about 20–40 minutes in gums, 40 minutes in nerve blocks, and a maximum of 3 hours in soft tissue. It is the most commonly used LA in pediatric patients because of its rapid onset and short dental procedures. It is available in 1:20,000 combination with levonordefrin which is a different profile compared to epinephrine. Their affinity to alpha and beta receptors is 75:25, a low effect on beta receptors. Levonordefrin is one-sixth of the potency of epinephrine. It has the lowest pKa of all LA used in dentistry, which explains the rapid onset [11]. Compared with lidocaine it has a potency of 2 and is metabolized thru oxidase in the liver, and renal elimination in 16% is unchanged.

4.3 Articaine

It is most recently introduced to dental practice. It is an amide type but possesses an ester group, making it the only hybrid LA, thus metabolizing in plasma as well as by the liver in which most of the process takes place, giving a result an inactive metabolite named articainic acid. It is equivalent to lidocaine in its vasodilation effect. It has a fast elimination time of approximately 27 minutes, compared to 40 minutes in amide anesthetics. It offers 90 minutes of pulpal anesthesia and 3 hours in soft tissue, being a reasonable choice in most dental procedures. This is available in dental cartridges at 4%, with epinephrine in 1:100,000 and 1:200,000 concentrations. The maximum dose is 7 mg/kg (72 mg per cartridge) for adults and 5 mg/kg for children under 15 kg [12]. It is the LA with higher risk of postoperative paresthesia in the jaw while rarely happening in other non-dental specialties such as orthopedic, spinal, or eye [13, 14].

4.4 Bupivacaine

It is a long-acting amide LA. Intrapulpal anesthesia as long as offers 6 hours and solf tissue up to 12 hours. It is also the most toxic drug, characterized by cardiovascular affection, inducing sudden collapse. In the same way it can induce longer duration of seizures which may be explained by its slower elimination (0.58 L/minute clearance) and a long half-life. It is four times more potent than lidocaine [15]. Although related to mepivacaine because of their molecular composition they differ chemically, it is 35 times more liposoluble, easily crossing the membrane in nerve cells, binding strongly at receptor sites with greater inactivation of sodium channels which make slow recovery, hence the saying that it is not easily uncoupled from the receptor.

Nancarrow et al. studied fatal doses in several LA: for lidocaine 30.8 ± 5.8 mg/kg/h, ropivacaine 7.3 ± 1.0 mg/kg, and bupivacaine 3.7 ± 1.1 mg/kg. Due to this finding, the toxicity threshold of bupivacaine in dental procedures is low and, an unnoticed injection during infiltration is dangerous and not recommended or approved by the FDA in children [16].

Bupivacaine is available in dental cartridges in 0.5% with 1:200,000. Ropivacaine has demonstrated a 75% safer profile than bupivacaine. It is a long-acting LA with the highest pKa (8.1) and binding to plasmatic proteins in 95% compared to the rest of the drugs used in dentistry, which gives a slowest onset, although in dental procedures this is usually not an issue. With a fast onset of 4-8 minutes after injection, 99% of patients reported low sensitivity in the lips vs. 100% reported with lidocaine [17]. The majority of reports of bupivacaine used in third molar extractions and root canal has confirmed its efficiency and security.

It is four times more toxic than lidocaine, so the use in dentistry is in lower doses than other areas in anesthesiology. A low incidence in paresthesia near 0.5% with this LA in combination with epinephrine at 1:200000 in an inferior alveolar nerve

block has been reported [18]. Chapman and Ganendran reported that patients blocked with bupivacaine and epinephrine for the third molar extractions did not need pain medication at 4 hours after surgery compared to the control group (lidocaine 2%) in which all of the patients were given pain medication at the same time. Seventy percent of the bupivacaine group received medication at 8 hours after surgery, versus 100% in the lidocaine 2% group [19].

4.5 Prilocaine

For dental anesthesia, it is available in 4% concentration with or without epinephrine 1:200,000. Prilocaine by itself induces more vasodilation than mepivacaine and less than lidocaine. It shares the same length of action, although a slower onset. Toxicity is lower than lidocaine. At doses above 600 mg, there is a higher risk of developing methemoglobinemia. In levels below 20%, clinical symptoms such as cyanosis, respiratory distress, and cardiovascular collapse are not seen. Mainstay treatment for methemoglobinemia is 1% methylene blue, 1–2 mg/kg intravenously slow 10-minute drip. Toxicity signs less severe than lidocaine. Prilocaine at 4% is contained in cream form commercially named EMLA (lidocaine-prilocaine) commonly used to produce skin numbness before any type of procedure such as phlebotomy; the downside is the time (30 minutes) to prepare the skin completely. The maximum recommended dose in adults is 600 mg and 400 mg in children. It may provide anesthesia up to 60 minutes in an inferior alveolar block or even 4 hours in soft tissue. It is important to remember that prilocaine is contraindicated in patients with congenital or idiopathic methemoglobinemia, heart failure, or chronic pulmonary disease.

5. Pharmacology of vasoconstrictors

The LAs commonly used in dental anesthesia are vasodilators, so they increase the blood flow in the injected site and perhaps enhance the concentration of the drug in blood and the probability of anesthetic overdose. The increase in blood flow also results in a short duration of action, although it depends on other factors, such as protein binding capacity.

Vasoconstrictors are adjuvant substances to LA that play an important role in dental anesthesia, producing deeper anesthesia and greater action duration, decreased systemic toxicity possibility, as well as bleeding reduction.

The most used vasoconstrictor in dental anesthesia is epinephrine, available in concentrations of 1:50,000; 1:100,000; and 1:200,000. It is rapidly metabolized by oxidation or conjugation, and its half-life is a few minutes, but its effects can last up to several hours. Malamed et al. showed in experimental animals that the application of 2% lidocaine with epinephrine 1:100,000 around the sciatic nerve reduces the blood flow in 79% (**Table 3**) [20]. Other vasoconstrictors used in this clinical setting are norepinephrine and levonordefrin [21]. Low plasmatic concentrations of adrenaline can raise heart rate, cardiac output, and systemic vasodilation because of its β1 adrenergic effect. The stimulation of adrenergic receptors alpha and beta occurs in 50/50 proportion; so that alpha-adrenergic stimulation causes peripheral vasoconstriction, while beta-adrenergic stimulation produces tachycardia. It is used to prolong the action time of LA, decrease dental bleeding, and improve the visibility of the surgical field. Its effect limits the diffusion of LA from the injection site and its systemic absorption reducing the possibility of systemic toxicity. Although, in general, vasoconstrictors are not contraindicated, the risk level depends on the characteristics of each patient; people with certain cardiac or endocrine diseases or

1:100,000 = 0.01 mg/mL or 10 μg/mL
1:200,000 = 0.05 mg/mL or 5 μg/mL
1:50,000 = 0.02 mg/mL or 20 μg/mL
1 epinephrine cartridge 1:200,000 = 9 μg/mL
1 epinephrine cartridge 1:100,000 = 18 μg/mL
1 epinephrine cartridge 1:50,000 = 36 μg/mL
1 levonordefrin cartridge 1:20,000 = 90 μg/mL

Table 3.
Vasoconstrictor concentrations in mg/mL.

taking medicines that affect the sympathetic nervous system have a greater risk of having deleterious side effects.

The LA with epinephrine for dental use are in the following proportions: 1:50,000 (0.02 mg/mL), 1:100,000 (0.01 mg/mL), or 1:200,000 (0.004 mg/mL) [22]. The administration of 2% lidocaine with epinephrine 1:100,000 (a cartridge) produces plasmatic concentration of 240 ± 69ρ g/mL. With the administration of three cartridges of epinephrine or 54 μg, the blood concentration is 302 ± 142 ρg/mL.

Usually, the administration of one cartridge of LA with epinephrine is not associated with cardiovascular changes, while the administration of three cartridges of LA with epinephrine at the same concentration is associated with five times the concentration of epinephrine in plasma, and with it there may be systemic cardiovascular alterations. However, it is not necessarily associated with the dose [23].

The umbral value of plasmatic epinephrine to develop hypertension is between 50 and 100 ρg/mL; the umbral value for systolic blood pressure is 75–125 ρg/mL, while the umbral value to rise the diastolic blood pressure is 150–200 ρg/mL. Barkin et al. found that 2% lidocaine 1:100,000 can produce non-serious cardiac arrhythmias in 16% of dental patients. The study does not specify with was the most frequent arrhythmia or if any treatment was administered [24].

Unfortunately, the vasoconstrictor effects are not always beneficial. In special situations vasoconstrictors can affect the patient; an example of this are the patients with limited cardiovascular systemic reserves, such angina pectoris, previous myocardial infarction, systemic hypertension, and non-controlled hypothyroidism. In such patients, epinephrine can produce indirectly central nervous system excitation, including systemic hypertension, tachycardia, tremors, headache, palpitations, cardiac arrhythmias, and stroke. Comorbidities recommendable to use lidocaine with epinephrine 1:100,000 to epinephrine dose of 0.04 mg = 40 μg as a total dose.

Procaine is a potent vasodilator and cannot produce adequate anesthesia if it is used without a vasoconstrictor. Lidocaine is also a vasodilator but has enough potency to be used alone. In contrast, mepivacaine has minor vasodilator properties and can be used with or without vasoconstrictors [25].

Norepinephrine as a vasoconstrictor is seldom used since fatalities due to hypertension have been reported. Another disadvantage at a local level is being a quarter less vasoconstrictor than epinephrine and having a shorter half-life.

6. Local and systemic complications of LA

Local anesthesia is the gold standard for surgical dental procedures; it has defined as a technique that produces loss of sensitivity, without losing

consciousness. Although anesthetics are defined as safe medications, some complications have been described.

The incidence of complications related to dental anesthesia is 4.5%, and the most common are needle break, paresthesia, transient facial paralysis, hematoma, toxicity and rarely allergy, dizziness 1.3%, tachycardia 1.1%, agitation 1.1%, nausea 0.8%, chills 0.7%, syncope, seizures, and bronchospasm [26].

As the same way, it can be complications related to additional vasoconstrictors of LA. It has been shown that the increases in plasmatic catecholamines observed after the LA infiltration are mainly due to higher doses of vasoconstrictors. Vasoconstrictors increase heart rate in 4.1% of patients and increase 20% with respect to baseline. Coronary insufficiency, arterial hypertension, myocardial infarction (in the last 6 months), congestive heart failure, pheochromocytoma, hyperthyroidism, and diabetes mellitus are risk factors for the use of vasoconstrictors during dental anesthesia [27].

Patients receiving LA are not always healthy; they can have hypertension, history of myocardial infarction, sequelae of diabetic neuropathy, cardiac disease, serious dental infections, are patients in extremes of life. Some are taking aspirin for various reasons: this drug inhibits the secretion of thromboxane, adenosine diphosphate, and serotonin, chemicals mediators necessary to the platelet plug formation. Therefore, all patients taking aspirin should be recommended to suppress it at least 6 or 7 days before the dental intervention. The aspirin binds to the platelet during its half-life of approximately 7 days, so by removing aspirin, the new platelets will function adequately. In arrhythmic patients the use of anesthetics with vasoconstrictors is not recommended.

More than 45% of dental patients have one or more concomitant disease in their medical history, and near 20% of patients will have some disease and drug or food allergy. The secondary effects of LA are mainly present in patients with risk factors; in such patients the secondary effects can raise to 5.7% [28], which may result in greater morbidity. Nowadays, although lidocaine is the most used LA in dentistry, there are other LAs such as articaine that could displace the use of lidocaine.

Complications of LA include local and systemic effects. Local complications include:

6.1 LA failure

The possibility that local dental anesthesia fails is remote. Timely identification of the reasons of regional anesthesia failure in dental or maxillofacial surgery is essential to adopt the measures required for its correction. Some factors of anesthetic failure involve bifid inferior alveolar nerve, retromolar foramen associated to accessory innervation, double or accessory mental foramen, the relation between the infiltration technique and bone density, accessory innervation in the case of the mylohyoid nerve and first cervical branches, cross innervation of the incisors, inactivity in the presence of tissue inflammation, inactive LA, incorrect technique, and subjective perception on the part of anxious patients [12]. **Table 4** lists other important factors.

6.2 Hematoma

Damage to blood vessels is usually caused by the tip needle, the blood accumulated inside the oral tissues, and the swelling located in any tissue which acts as an irritant of the tissue and causes pain and trismus. Accumulated blood can

1. Inadequate anatomical selection
2. Insufficient LA dose
3. Insufficient time for the LA diffusion
4. Administration of LA in swelling or infected tissue
5. Use of expired LA

Table 4.
LA failure in dental practice.

be a culture medium for oral bacteria, especially in diabetic patients or those with immune deficiency.

6.3 Nerve damage

The needle can damage a nerve which produces a partial or complete deficit with motor or sensory abnormalities that usually have a full recovery.

6.4 Transient facial nerve paralysis

This complication is caused by the introduction of LA into the parotid gland capsule, which is located at the back of the edge of the mandibular branch. If the LA is deposited on this site, a transient paralysis of muscles of the face occurs. The LA is applied next to the facial nerve, so the motor blockade would cause a temporary paralysis of the muscles of the face; clinically a modification in the face expression appears. The duration of motor paralysis lasts between 3 and 5 hours, and treatment is not required. An important clinical situation is that patient cannot close the affected eye and is necessary to avoid the dry eye during this period of involvement [29].

6.5 Needle rupture

Since the introduction of non-reusable needles in dental anesthesia, needle rupture has been an extremely rare complication. Progrel et al. estimated this risk at 1:14 million, more specifically in the case of inferior alveolar nerve block. In the analysis of broken needles, it was found that the majority were short or G30 ultra-short needles (20 and 10 mm). The inferior alveolar nerve block was the most frequent in 79% of the cases and the alveolar superior posterior nerve in the rest of the cases. Additional factors are pre-bending of the needle before injection, the unexpected and sudden movement of the patient at the time the needle is entering the soft tissue, and strong contact of the needle with the bone [30].

6.6 Systemic complications of LA

Local anesthetic-related systemic complications are associated with the nature of the drug and/or their composition. Systemic complications are as followed:

6.6.1 Systemic or local infection

Spreading the potentially dangerous infection within mouth soft tissue to neck or head may be caused by needle trauma. Dental abscess is a great danger to

patient's health and a high risk for airway management by the anesthesiologist due to the possibility of rupturing it during the intubation. It is important to emphasize that LA should not be applied in infected soft tissue.

Bacterial endocarditis is not a complication related to the use of LA per se; however it can be related as a post procedure bacteremia after any type of injection to the mouth. Dental surgery that involves mucosa o contaminated tissue such as a molar extraction can produce a transient bacteremia and facilitate infections at a distance, especially in cardiac valves or endocardium. The most common bacteria is hemolytic streptococcus viridians. Patients with dentures can develop bacteremia from gum ulcers or gingivitis. Numerous studies have shown a possible odontology-related etiology of bacterial endocarditis in up to 20% of the patients. Although prophylactic antibiotics are a commonly accepted practice, the American Heart Association (AHA) in 1997 described multiple prophylactic strategies: (a) amoxicillin 2 g, 1 hour prior to the treatment, and (b) clindamycin 600 mg, 1 hour prior to treatment [31].

6.7 Cardiovascular manifestations

Heart complications related to dental procedures may increase up to a 5.7% in patients with identified risk factors. Patients with coronary heart disease, cardiac surgery, or heart failure show greater plasmatic lidocaine levels; therefore a 50% reduction in maximum dosage of LA is recommended. Potassium levels and acidosis may worsen the adverse effects in the myocardium. High-risk patients should be limited to 30-minute dental surgery; after that time complications may rise up to 15% [32].

Cardiovascular collapse is described as the most severe LA complication, associated with high mortality. It is produced by intravascular injection of AL and is manifested by arrhythmias, heart failure, and arterial hypotension that can end in death if not treated in a timely manner. Usually, the doses of LA used in dentistry rarely exceed the limits to cause cardiovascular problems, although in exceptional cases, small amounts of AL may be capable of cardiac arrest [33]. For more information, we refer the reader to the chapter on LA systemic toxicity included in this book.

6.8 Local anesthetic overdose

Most overdose reactions occur during LA injection or within the next 5–10 minutes. Clinical symptoms involving the CNS are, for example, generalized numbness, facial numbing, anxiety, restlessness, confusion, chills, seizures, or respiratory arrest [7]. A simple way to avoid LA injection into the blood vessels is to aspirate before and during the injection. Systemic toxicity depends on several factors such as speed of injection, site, and combination with vasoconstrictors. For example, maximum dose of LA in a pediatric patient may be mistaken with an adult and cause an overdose. High concentrations of LA articaine and prilocaine may exacerbate and overdose. Serum concentration of LA less than 5 μg/mL produce moderate sedation and analgesia, but at concentrations as high as 5–10 μg/mL, it can cause incoherent talk, dysphoria, diplopia, muscle contractions, or seizures [34].

6.9 Plasma cholinesterase deficiency

Esther-type LA should be avoided in patients that may carry this rare enzyme deficiency, due to the metabolism of this anesthetic. Methemoglobinemia is a rare complication associated with excessive metabolites of certain LA, mainly prilocaine, causing oxidation of the ferrous component in the blood to a ferric

form, and poorly delivered oxygen to tissues commonly expressed as hypoxia. Unique features, such as a saturation gap and chocolate-brown-colored blood, can raise suspicion for methemoglobinemia. The use of articaine and benzocaine have also been associated with methemoglobinemia and manifests with cyanosis that does not respond with supplemental oxygen. When high methemoglobinemia levels are present, clinical symptoms such as nausea, sedation, seizures, and coma may appear. Treatment is with methylene blue. Thus, patients with congenital or acquired plasma cholinesterase deficiency should avoid exposure to these LAs [34].

6.10 Allergic reactions

These reactions are extremely rare in dentistry. The most common allergic reactions are allergies to latex, acrylates, and formaldehyde. While polymethylmethacrylate and latex trigger delayed hypersensitivity reactions, sodium metabisulphite and nickel cause immediate reactions. Most adverse reactions are caused by systemic complications or anxiety as a result of pain or needles, generating hyperventilation or syncope that may be confused with a faulty allergic reaction. True allergic reactions are caused mainly by ester LA. They are not dose related. Only 0.7–1% of all allergies are authentic hypersensitivity reactions and caused after administering LA [35]. It is fundamental to have previous contact with the allergen, and then a usual latency period occurs until a second exposure. Hypersensitivity reactions related to LA are classified into two types: type I reactions or humoral are immediate and severe such as anaphylactic shock, angioedema, fever, and photo sensibility. Type IV or cellular are delayed and manifested through moderate dermatologic reactions such as hives or cutaneous rash. Anaphylactic shock usually occurs within a short exposure to the antigen, and symptoms include cardiovascular collapse in 76.3%, bronchospasm in 44.2%, and skin in 69.9%. Cardiovascular collapse presents as abrupt drop in blood pressure, bradycardia, and desaturation followed by bronchospasm and redness in thoracic area and face are common. Initial management is epinephrine 1-5 mcg/kg [36]. Intramuscular application, fluid bolus, secured airway, and increase in oxygen concentration. There is a frequently reported contact dermatitis in healthcare workers that are exposed to parabens and bisulfates used as preservatives in local anesthetic preparations which can cause an allergic reaction [37, 38].

6.11 Drug interactions

There are some well-known interactions with LA, such as tricyclic antidepressants and beta blockers. The first act is by inhibiting the catecholamine reuptake, thus increasing the concentration at the presynaptic sympathetic binding site. In patients taking this kind of medication, it is recommended to limit the amount of epinephrine to a maximum of 0.05 mg/dose. Beta blockers, on the other hand, inhibit arteriolar vasodilation effect of drugs as epinephrine in combination with LA, which increase the predominant vasoconstrictor alpha adrenergic effect. The end result could be an increase of arterial blood pressure and sympathetic effects. Another reported interaction is the diminished metabolism of amide LA. Sedatives, opioids, opioids, antihistamines, magnesium sulfate, and LA may increase CNS depression and respiratory drive which has to be titrated with caution. LA and some antiarrhythmic like quinidine may increase myocardial depression. Antimyasthenic agents such as neostigmine could antagonize the effect or muscle contraction. Anticholinesterase lowers the metabolism of ester LA. Ester LA such as procaine combined with sulfas could inhibit antimicrobial action [34].

7. Conclusions

Anxiety, stress, and pain are very frequent characteristics in dentistry. Dental anxiety can be considered as a universal phenomenon with a high prevalence, being one of the main causes of medical emergencies in the dental office, so its prevention is an essential part of patient safety and quality of care. The LA used in this clinical setting must be selective in nerve tissue; be powerful enough to produce complete anesthesia without tissue damage; have a reversible action within a predictable time; have minimal side effects and, also, reduced systemic toxicity, and few hypersensitivity reactions; have a short latency period with the duration of the effect adaptable to the desired; not cause pain during injection; be compatible with other components in the solution and not easily modified by sterilization processes; not be sensitive to variations in pH; be stable in the solution; and have sufficient penetration. Lidocaine and articaine with epinephrine are the most used, although mepivacaine and prilocaine are still options. It is mandatory to monitor side reactions, especially systemic toxicity.

Author details

Enrique Hernández-Cortez[1]*, Cecilia G. Sandoval Larios[2] and Juan Carlos Flores-Carrillo[3]

1 Department of Anesthesia, Federación Mexicana de Colegios de Anestesiología, A.C., León, México

2 Anesthesia and Cardiovascular Anesthesia, Airway Committee Federación Mexicana de Colegios de Anestesiologia AC, Hospital Angeles León, México

3 Anesthesia and Critical Care Medicine, Hospital Angeles Tijuana, México

*Address all correspondence to: kikinhedz@gmail.com

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